ICP, Cerebral Edema, and Brain Bleeds

Introduction

This lesson is about increased intracranial pressure (ICP) in general, treating increased ICP due to any cause, and select causes of increased ICP—cerebral edema and brain bleed. Infections—especially abscesses and cryptococcal meningitis—and brain tumors are the leading causes of increased ICP. Infection was covered in Microbiology, and brain tumors get their own lesson later in the Neuroscience module. Finally, hydrocephalus (covered in Neuroscience: Cortex #2: *The Normal CNS: Cells, Fascicles, and Meninges*) can also cause increased ICP.

We start out with the pathophysiology and symptoms of ICP, review some treatments, and then go into some different causes of increased ICP.

Intracranial Pressure

We focus on systemic blood pressure control and its relationship to ICP in Cardiac: Hemodynamics #4: *Blood Pressure Regulation*. We review that information here, then expand on the information to include the symptoms of increased ICP and the teleology behind it.

The cerebral perfusion pressure (CPP) is dependent on the perfusion pressure into the blood vessels of the cranium (MAP) and the collapsing pressure exerted upon those blood vessels (ICP). The brain is within a rigid skull, within the cranial cavity. Normally, there are blood vessels, lymphatics, and nerves within the cranial cavity, within the subarachnoid space, and within the parenchyma. Any extra space is filled up with cerebrospinal fluid (CSF). There is an ICP all the time; it is the driving force that pushes CSF towards the arachnoid granulations. Normal ICP is 5–15 mmHg. Normal MAP is approximately 90 mmHg. The ICP adds resistance to the blood vessels, but the MAP overcomes that resistance. Thus, CPP is determined by both the MAP (perfusion) and the ICP (resistance).

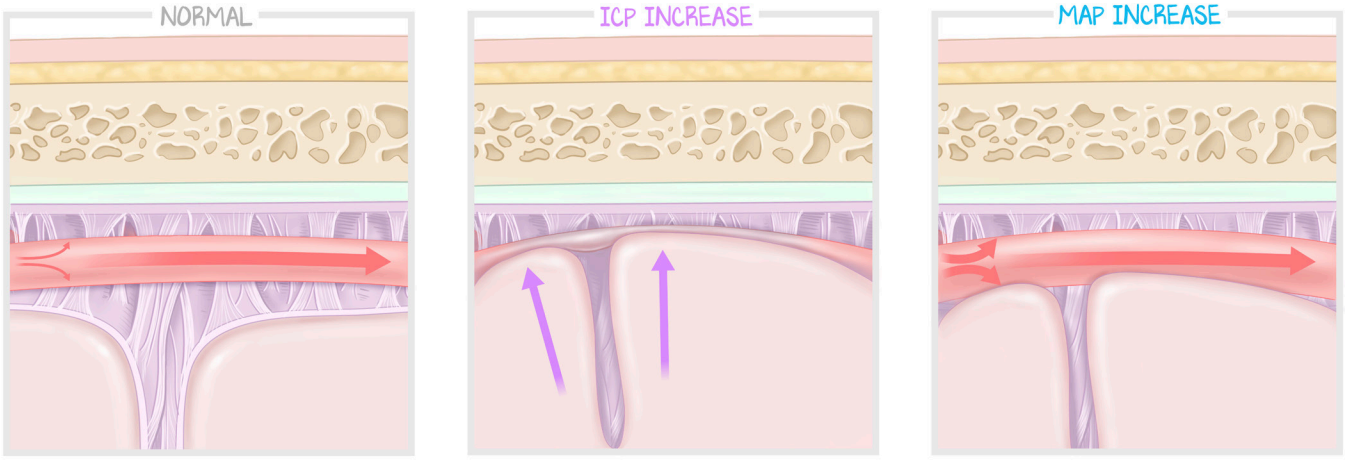

Figure 4.1: Cerebral Perfusion Pressure
Because the brain is within a rigid skull with limited space, there is a pressure—the ICP—exerted on its blood vessels. Normally, the MAP is greater than the ICP and keeps the vessels open. If the ICP rose and MAP did not, the ICP would collapse the vessels and reduce the perfusion to nil. If the MAP were to then increase, it would reopen the blood vessels.

If the pressure builds in the cranial cavity, as the **ICP rises**, the **CPP falls**. It falls because the intracerebral vessels are compressed by the increased ICP. To open those compressed vessels, there needs to be a stronger force pushing blood in. Thus, the brain instructs the cardiovascular system to **increase**

MAP to maintain cerebral perfusion. It induces vasoconstriction, tachycardia, and an increased force of contraction. The **baroreceptors—**those located in the aortic arch, at the bifurcation of the right common carotid and the bifurcation of the left common carotid—sense this increased pressure. When they do, they tell the medulla to inhibit sympathetic discharges and stimulate the parasympathetics. The two signals compete. The medulla predominates in the heart, resulting in bradycardia. The brain predominates in the blood vessels, leading to hypertension. **Bradycardia and severe hypertension** are two of the three elements of Cushing's triad, products of Cushing's reflex, and are signs of increased ICP.

The main sign of increased ICP is **headache**. The headache is **worse in the morning** and with actions that increase ICP (**cough**, **sneeze**, and **Valsalva maneuver**). The morning headache is a product of lying flat, which evenly distributes the MAP across the body, resulting in higher ICP, higher CPP, and greater perfusion (perfusion of the brain is highest when asleep) than when in an upright position, which enables gravity to reduce perfusion pressure and facilitate venous drainage with its downward pull. It isn't the recumbency; it's what gravity does to perfusion pressure to the brain. To drive this point home, hanging upside-down would hurt the most. **Projectile vomiting** without nausea is another sign of increased ICP. **Papilledema** is a result of the eyes being outside of the skull. The foramen through which the vessels of the eye exit the skull into the orbit both acts as a release valve and may have difficulty getting blood drained (i.e., back into the cranium) if the pressure is high. Less helpful signs are a **"nonlocalizing sixth"** (cranial nerve VI palsy, inability to abduct an eye past midline), which is present in many pathologies, and **seizures**, which have many etiologies.

Diagnosing the etiology of increased ICP is done with **brain imaging** (CT or MRI; you will not have to choose at this stage of training). Increased ICP can be confirmed with a **lumbar puncture** with an opening pressure. Do not perform a lumbar puncture until the imaging is completed, and a space-occupying lesion (tumor, abscess, bleed) has been ruled out. Performing a lumbar puncture when a lesion is present may result in herniation of the brainstem through the foramen magnum. The pressure is increased, and all the structures inside the cranium feel it and are looking for a way out. Because the CSF is an intact system, alleviating the pressure by draining the CSF at the L-spine would create a vacuum, pulling all the structures down towards it. Fluid leaves, but so too do the cerebellar tonsils, resulting in herniation syndromes. Other times, such as in hydrocephalus, relief of the pressure may reduce symptoms.

Treatment of Increased ICP

The brain increases MAP to maintain CPP. It does so at the cost of every other organ. Relief from the underlying cause is curative. In some instances, the patient is either too unstable or the cause not easily cured, in which case you can do some symptom-relieving maneuvers.

Raising the head of the bed will force the heart to pump against gravity and facilitate the drainage of venous blood and lymphatics. You never perform neurosurgery sitting up because the venous drainage is at negative pressure, and opening a vein will cause air to get sucked in. Patients who need this intervention might get neurosurgery shortly, but until the surgery begins, lowering the perfusion pressure and facilitating venous drainage lowers ICP.

Hyperventilation will blow off CO_2. The brain is responsible for regulating CO_2, using CO_2 as a surrogate for serum pH (the details of this are covered in Pulmonary: Lung #2*: Mechanics and Regulation of Respiration)*. When the CO_2 goes up, the respiratory rate increases to eliminate ("blow off") more CO_2; when the CO_2 goes down, the respiratory rate decreases to retain CO_2. That is CO_2 and the medulla from a pulmonary perspective. But CO_2 has an effect on autoregulation, on the set point of the arteriolar tone. Decreased CO_2 will lead to **vasoconstriction** of the cerebral arteries. Vasoconstriction will locally increase resistance and decrease flow, thereby reducing perfusion pressure in the brain. It

does also compromise perfusion, but if the person is in a state where there is such a high ICP that intervention is warranted, that intervention will likely happen soon, and hyperventilation temporizes the patient in the meantime.

Mannitol, an osmotic diuretic, is best used in the treatment of cerebral edema-induced intracranial hypertension. The problem is excess fluid within the parenchyma, between cells. Diuretics drain the whole patient of fluid by facilitating the elimination of fluid via the kidneys. As fluid leaves the bloodstream and into the kidneys, fluid shifts draws fluid out of tissues into the bloodstream. That is how all diuretics work. Unlike other diuretics, mannitol does more than just enable volume shifts through diuresis. In addition to the whole body fluid shifts, mannitol is osmotically active, and when in the cerebral vasculature, will favor the local shift of extracellular fluid into the bloodstream and out of the brain.

Craniotomy (-tomy means *cut* a hole) is the drilling of a hole in the skull to relieve pressure. This is an emergent procedure that is especially useful for the relief of subdural hemorrhage. As the hemorrhage continues and the hematoma expands, there will be more and more pressure. Drilling a hole over the bleed will alleviate the pressure of the hemorrhage, and blood will come out the hole instead of remaining trapped in the cranium. It isn't as effective for other causes of increased ICP because if the problem doesn't come through the hole, the brain will, and that is a herniation.

Decompressive craniectomy (-ectomy means to *cut out* or remove) is the most invasive. It is the surgical removal of part of the skull. This procedure both enables a neurosurgeon to operate on a defect if one is visible and buys time. With the skull removed, the brain can "herniate" through the opening. But because the opening was made intentionally large, with clean edges, it simply acts as a release valve rather than causes injury. Of course, with the skull removed, infection is a major complication risk, and the wound needs to be meticulously cared for.

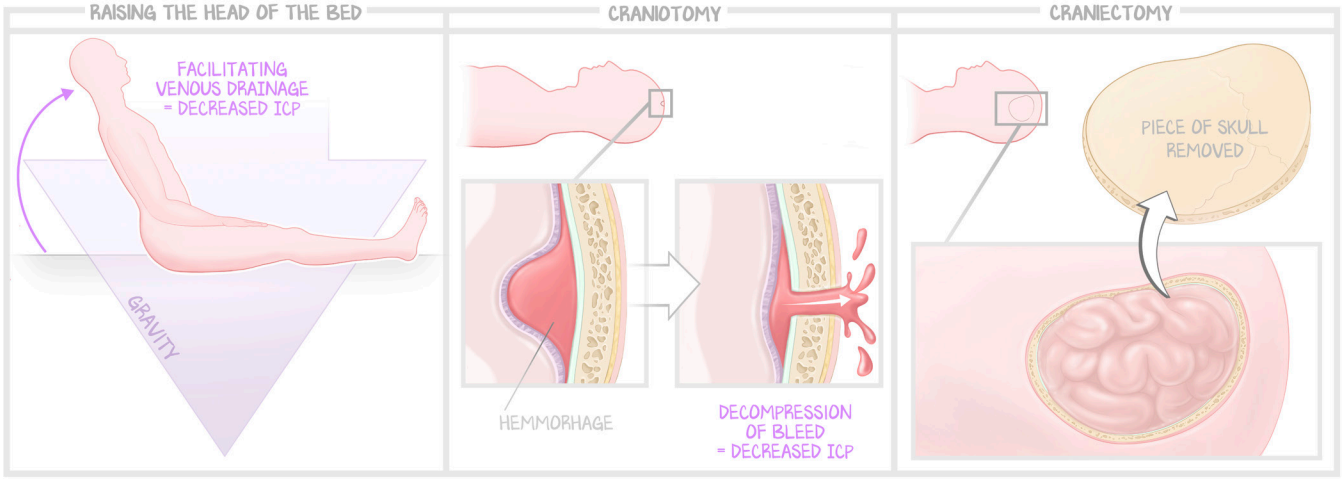

Figure 4.2: Treating Increased ICP
Raising the head of the bed increases gravitational pull, which reduces blood in and facilitates blood out. A craniotomy can relieve the pressure of a hemorrhage by providing an escape allowing that hemorrhage to leave through a hole in the skull. Craniectomy exposes the meninges to the atmosphere, but allows for a release valve of the pressure.

You won't have to decide which thing to do in Basic Sciences. Anyone with evidence of increased ICP gets *"medical therapy; elevate the head and hyperventilate."* For cerebral edema, use mannitol. Surgical interventions include removing part of the skull (all causes of ICP benefit) and drilling a hole (only for brain bleed).

The Outcome of Increased ICP = Herniations

A herniation is the displacement of brain tissue past a rigid dural fold (the cerebral falx or the tentorium cerebelli) or through openings in the skull (the foramen magnum) because of increased ICP. Initially, structures are moved around by an increased ICP. Because grey matter is neuron cell bodies and glial cells, and white matter is neuron axons and glial cells, they have mass and take up volume. The brain parenchyma may be squishy compared to other organs, but it is tougher than ventricles and veins. So when ICP increases, when something other than the-brain-and-its-CSF shoves on the-brain-and-its-CSF, everything moves, but the CSF moves the most. The ventricles will show signs of **midline shift**. The ventricles will be deformed and smaller, giving way to the pressure. As the pressure gets even higher, the next to go are the veins and sinuses. These changes are not usually visible on imaging, as the thing causing the increased ICP and the midline shift is so obvious. Eventually, the ICP becomes so high that it displaces the brain itself. The displaced tissue is then compressed against a rigid structure, such as a dural fold or the skull, severely compromising the displaced tissue's function. In addition, the displaced tissue may also compress the arterial supply of nearby structures, inducing ischemia and further exacerbating cerebral edema and ICP.

Without intervention, ICP so severe as to cause herniation is fatal. The location of the initial pressure will determine what herniates, and therefore what the herniation syndrome will be. You are responsible for identifying three syndromes, explained next.

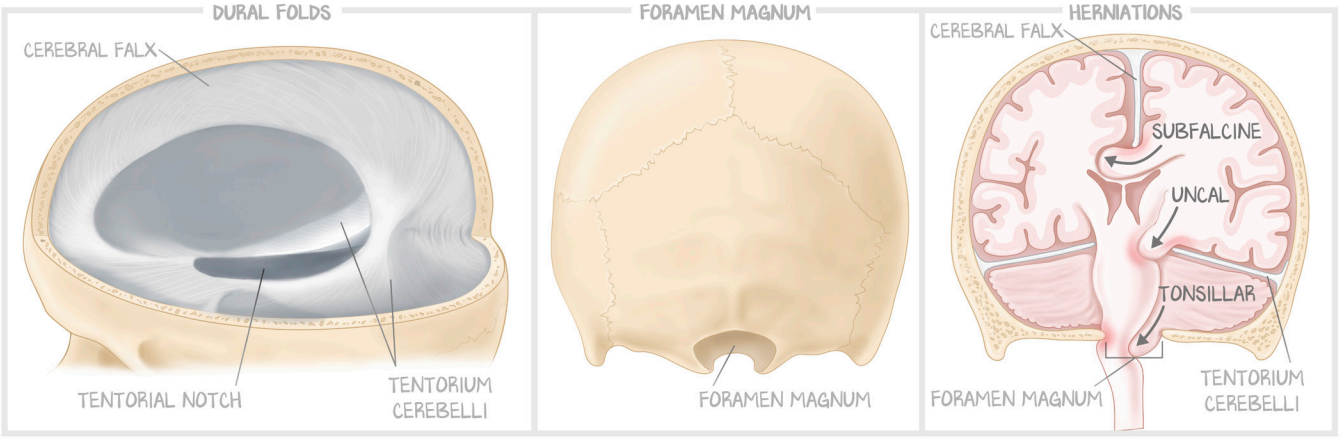

Figure 4.3: The Anatomy of Brain Herniation
The cerebral falx separates the left and right hemispheres. The two tentorium cerebelli separate the cerebellum from the cortex on either side. The tentorial notch is the space occupied by the brainstem and lies between the tentorium cerebelli. The foramen magnum is the opening in the skull through which the brainstem exits. The three herniations to learn are subfalcine, uncal, and tonsillar.

Subfalcine herniations are when the **cingulate gyrus** (of the limbic system) herniates under the cerebral falx. The inferior margin of the cerebral falx traces along the corpus callosum. The cingulate gyrus is displaced away from the side of the lesion. The cingulate gyrus is the most inferior structure above the corpus callosum. Thus, when the brain is forced laterally, the cingulate gyrus is forced laterally and slips under the cerebral falx. The **anterior cerebral artery** runs along the top of the corpus callosum, feeding the medial frontal lobe and parietal lobe. Thus, subfalcine herniations present with increased ICP and stroke symptoms of the **anterior cerebral arteries** (Motor and Sensory Tracts #4: *Cerebral Vasculature and Strokes*).

Uncal herniations are when the **medial aspect of the temporal lobe** (the uncus) herniates under the tentorium cerebelli. The compression of the uncus is not what causes symptoms. Instead, the herniation's compression of other structures below the tentorium produces the classic findings. Compression

of the ipsilateral oculomotor nerve (CN III) results in **ipsilateral pupil dilation** (parasympathetic motor fibers) and **extraocular muscle paralysis** (CN III innervates four of the six muscles). As the herniation progresses (visualize the laterality), the uncus displaces the brainstem away from the lesion. Compression of the brainstem against the contralateral tentorium cerebelli crushes the axons of sensory nerve fibers (which just so happen to be the most lateral axons). Loss of the axons here will cause sensory defects contralateral to the axons damaged, which is ipsilateral to the side of herniation (you'll get rigorous training on this in the Motor and Sensory Tracts lessons). That means there will be a **loss of bodily sensation on the entire ipsilateral side** (hemianesthesia).

Tonsillar herniation is the herniation of the **cerebellar tonsils** through the **foramen magnum.** Much like uncal herniation, the herniation isn't the problem. The problem is that the herniation pushes the contralateral medulla up against bone. Compression of the medulla results in respiratory and cardiovascular arrest. It is a rapidly fatal herniation.

Central hernias are caused by diffuse cerebral edema. Often in patients with significant trauma, the progression of expanding edema force the top of the brainstem and posterior cerebrum through the tentorial notch. This shears blood vessels and compresses the eye nerves and nuclei bilaterally, resulting in **bilateral pupil dilation** (because of parasympathetic loss) and **bilateral extraocular palsies**.

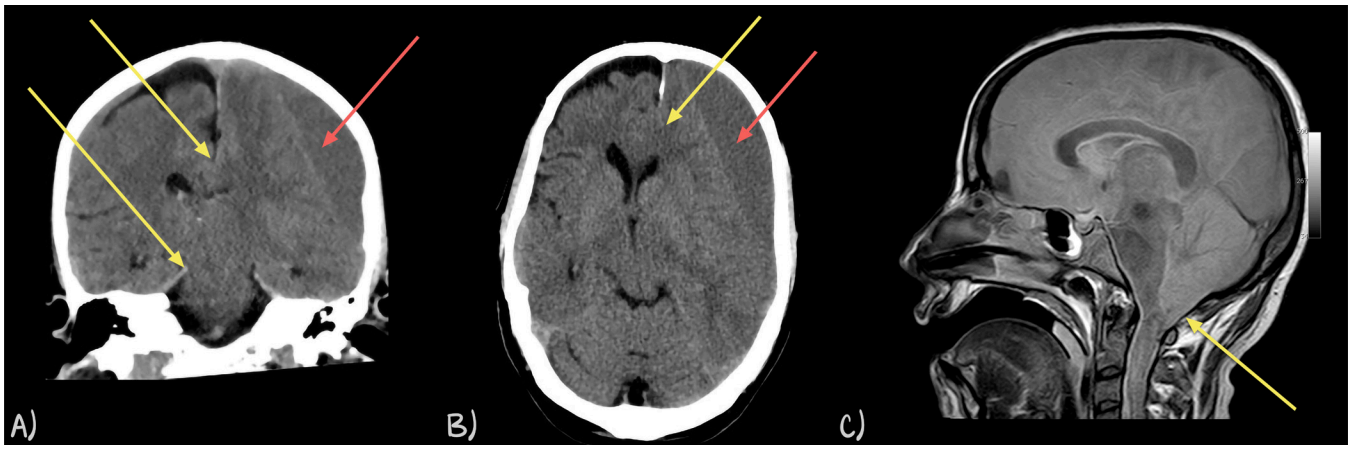

Figure 4.4: Visualizing Herniations
(a) Brain bleed (red arrow) causing a midline shift. You can see both a subfalcine herniation from left to right and an uncal herniation displacing the brainstem into the contralateral tentorium. (b) From the same patient as in (a), this transverse slice shows the collapse of the right ventricle, and even how the left cerebrum has been pulled down (extra CSF anterior) as the hernia progressed. (c) Sagittal MRI demonstrating the herniation of the inferior cerebellar tonsil down into the foramen magnum.

The Cause of Increased ICP #1: Cerebral Edema

Cerebral edema is excess fluid in the brain parenchyma. This is a microscopic addition of fluid that can be either localized (adjacent to an infection, infarction, or tumor) or generalized. It adds volume to the brain, causing swelling, which can lead to increased ICP. There are four types: vasogenic, cytotoxic, hydrocephalic, and osmotic.

Vasogenic edema is the result of a space-occupying lesion—abscess or neoplasm. The presence of something that isn't supposed to be there induces the immune system to respond. The astrocytes still try to do their job, but the endothelial cells are responding to inflammation the way they are supposed to—they dilate to allow for venous stagnation so leukocytes can get out of the vessels. And although it may be an appropriate response to the pathology in the brain, it allows for the effusion of fluid along with those immune cells, inducing cerebral edema. But it matters so much in the brain (as opposed to everywhere

else) because the brain is encased in the skull, and the edema can lead to increased ICP. Radiologically, this will affect the **white matter only** and will be "**finger-like**"—not in an obvious territory.

Cytotoxic edema is caused by infarction. Ischemia leads to failure of the Na^+/K^+-ATPase, accumulation of sodium in the cytoplasm, and subsequent cellular swelling. The swelling of cells (intracellular at first, opposed to extracellular as in vasogenic edema) will end with necrosis. The radiological appearance is often **wedge-like**, involving the grey and white matter (**blurring of the grey-white junction**), and will be in a **vascular distribution**.

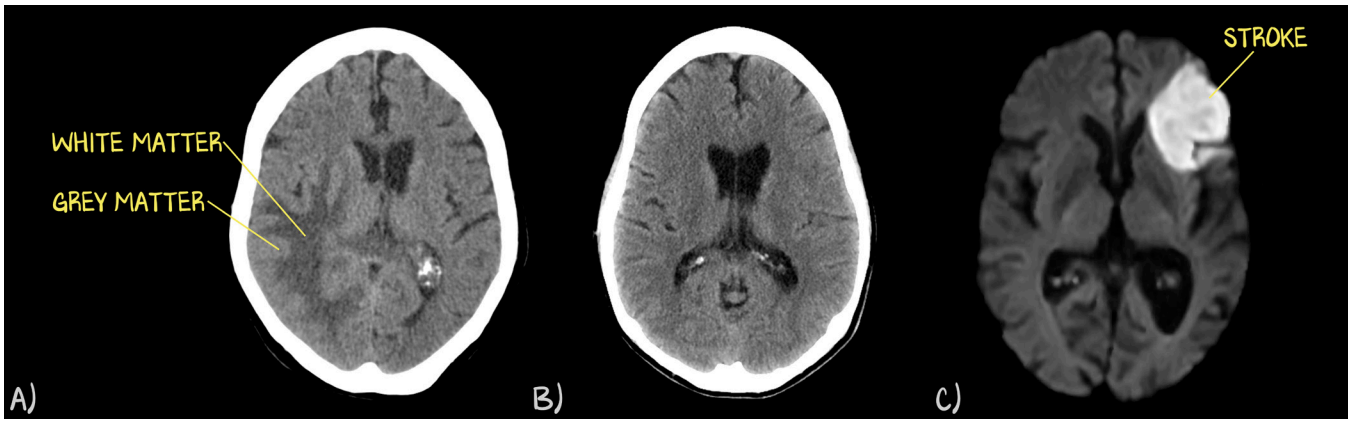

Figure 4.5: Visualizing Edema
(a) Vasogenic edema, sparing the grey matter on the edges of the skull. The lesion is dark grey and sends finger-like projections into the surrounding brain. The lesion is on the lower left. (b) CT of a normal brain for comparison. Grey and white matter are discernable, the ventricles are mostly even, and there are no shifts. (c) MRI of a left watershed-area stroke. In this phase of MRI, the lesion appears bright white. There is a loss of the grey-white junction within the lesion.

In truth, in clinical practice, both vasogenic and cytotoxic edema occur concurrently. However, they can be separated radiologically by the presence of a space-occupying lesion and involvement of the grey-white junction.

Hydrocephalic edema (formerly interstitial edema) occurs **around the lateral ventricles** when there is an acute obstruction of flow of the CSF. This is seen in **obstructive hydrocephalus**, where the ventricles become dilated due to increasing pressure. The hydrostatic pressure within the ventricles causes fluid to accumulate around the ventricles. This is a type of cerebral edema, but it is the hydrocephalus that predominates in causing the syndrome of increased ICP. If the pressure within the ventricles is such that it induces periventricular edema, the pressure exerted on the brain is high enough to cause symptoms. In this case, fixing the hydrocephalus will fix not only the edema but also the ICPs. We finish this lesson with the specific treatment of hydrocephalic ICP.

Osmotic edema is usually iatrogenic. Water follows osmoles—be they salt or glucose. When the blood is dilute, water will move into cells to balance the osmolarity. This will cause cellular swelling. This may cause symptoms of brain dysfunction but won't cause increased ICP. Severe hyponatremia may cause altered mental status and seizures. When the blood is concentrated, water will move out of cells to balance the osmolarity. This will cause cellular shrinkage. This may cause symptoms of brain dysfunction but won't cause increased ICP. Severe hyperglycemia may cause altered mental status and seizure. See the parallels? If you fix the glucose or sodium too quickly, the reverse fluid shift will occur. And, being abrupt, the shift will cause more problems than the original state. We're being vague here because osmolarity shifts are covered in the lessons about these subjects. We just want you to see that if a cell is shrunken and water shifts in too quickly (cerebral edema), the cell might die, and that if a cell is already swollen (cerebral edema) and water shifts out too quickly, the cell might die. The point is: do not fix blood osmolarity quickly.

The Causes of Increased ICP #2: Brain Bleeds

The skull encases the brain and meninges. Beneath the skull is the dura mater, the inelastic fibrous tissue that houses the dural sinuses. Beneath the dura mater are the leptomeninges, which comprise the arachnoid layer (7–10 fibroblasts thick, stem cells for the subarachnoid space's trabeculae and the pia matter's epithelium) and pia mater that form a basement membrane used by the astrocytes to form their glia limitans. Beneath the glia limitans is brain parenchyma. Small arteries and bridging veins travel within the subarachnoid space, separated from the CSF by cells of the arachnoid layer. Large arteries enter the skull through foramina and travel within the dura mater or the subarachnoid space of dural sinuses. We are going to use this information to predict which type of event results in which type of hemorrhage, and what different hemorrhages will look like on CT. The last piece of information you need is that on CT, bone is white, and blood is white.

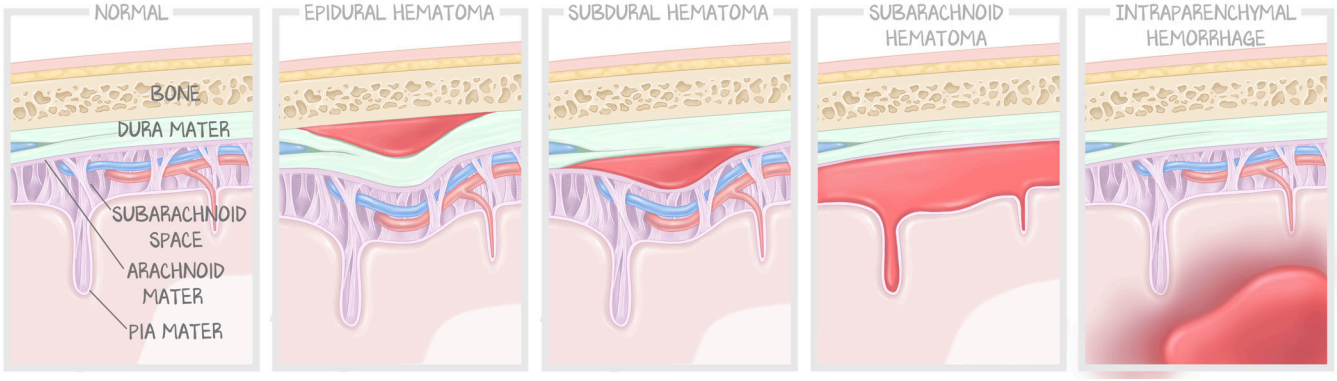

Figure 4.6: The Layers and the Bleeds
Normal anatomy of the meningeal layers and their relationship to one another. The remainder demonstrates what is happening in each bleed. Refer to this illustration as you read, and compare it to the imaging that follows.

Remember, the signs of ICP in general are agnostic of the cause. All brain bleeds, if they continue, will increase the ICP. We will discuss the unique features—mechanism, clinical course, and findings on brain imaging—that enable you to separate them from one another. There are four brain bleeds: epidural hematoma, subdural hematoma, subarachnoid hematoma, and intraparenchymal hemorrhage.

An **epidural hematoma** is caused by severe trauma, one that could fracture the skull or shear the large arteries. Most often, this is caused by damage to the **middle meningeal artery** due to strong, blunt force trauma to the side of the head. This is trauma of the skiing accident kind, or the baseball player taking a ball to the side of the head without a helmet. The space between the periosteum of the bone and the periosteal edge of the dura mater is a **potential space**. When a large artery bleeds, the blood wedges between the bone and the dura mater. The dura mater is attached to the bone at the bone's edge, at the fissures. When a large artery starts bleeding, it forces the dura mater away from the bone. The skull is sturdier than the dura mater, so the dura mater bows out from the bone. The bleed is well confined by the dura mater, so the brain imaging will show a **linear contour** and an **elliptical shape**. This is the "walk, talk, then die" syndrome. The patient will have a trauma, with either a loss of consciousness or not, but after a brief **lucid interval** (walk, talk), there is a **rapid progression** to coma and death (then die).

A **subdural hematoma** is a bleed (hematoma) below the dura mater (subdural) and above the arachnoid layer. This, too, is hemorrhage into a **potential space**. Although discontinuous, there is not supposed to be anything between the bottom of the dura mater and the top of the arachnoid layer. The bleed is between two layers and, like epidural hematoma, the weaker of the two layers bends to the force of the hemorrhage. This time, the arachnoid layer, a flimsy 7–10 cells thick, gives way while the dura

mater retains its normal shape, and the hemorrhage pushes the arachnoid layer towards the brain. The subarachnoid space is even flimsier, filled with CSF. But the blood is above the arachnoid layer, so it does not enter the subarachnoid space. If blood cannot get into the subarachnoid space, blood **will not appear in the sulci**. The arachnoid is a flimsy layer, and the subarachnoid no sturdier than the CSF within it, so the hematoma may appear to conform to the shape of the brain. Because there are no suture lines below the dura mater, this type of hematoma can continue to spread along the surface of the brain, giving it a **concave shape**. The vessels to associate with subdural hematomas are the **bridging veins**. Shearing of bridging veins is seen in two patient populations: children and the elderly. For adults, if the brain atrophies, the space it occupied fills with CSF. Alcohol abuse, Alzheimer's dementia, even the normal process of aging, all cause atrophy. As the brain shrinks away from the dura, these veins become stretched and are easily sheared, so subdural hematomas may occur after a **fall from standing** in **elderly** patients. These patients present with an **insidious headache** progressing to **encephalopathy** (often mistaken as dementia). In children, a subdural hematoma represents child abuse. Although their brains are robust and their bridging veins are not stretched (a fall from standing in a child will obviously not cause a subdural), if shaken vigorously by an adult, these veins can shear. Kids who are shaken this vigorously present **acutely,** often obtunded.

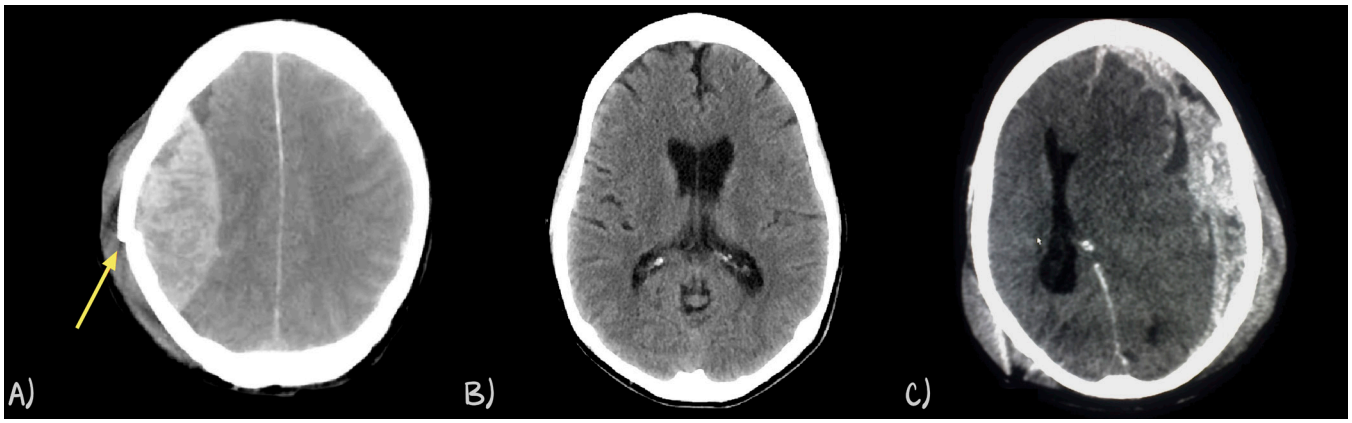

Figure 4.7: Visualizing Brain Bleeds, Part 1
(a) An epidural hematoma secondary to fracture (red arrow). The concave shape on the outside of the skull is a hematoma in the side of the head. You can see the white ellipse pushing on the brain, and pulling the top, unaffected brain, away from the wall. (b) CT of normal brain for comparison. Although this image is from lower in the brain (the lateral ventricles are visible), it certainly shows that the white lens-shaped bleed is abnormal. (c) A subdural hematoma. Notice its concave shape, conforming to the shape of the brain beneath it. At the top, it appears to fill the sulci, which would make it a subarachnoid hemorrhage. But look at the bulk of the bleed, especially at the 2 o'clock position, where there is black between the grey brain and white bleed. Also, notice the obliteration of the ipsilateral ventricle and midline shift (implying subfalcine herniation).

Hemorrhages in the epidural or subdural space are typically associated with trauma, as above. Hemorrhages in the subarachnoid space or the brain parenchyma, in contrast, are more often a manifestation of underlying medical disease.

Subarachnoid hematomas are usually nontraumatic. Although it is possible to get these bleeds from trauma, we teach them as being secondary to medical disease—either **hypertensive emergency** or a **rupture of a berry aneurysm**. These are medium-to-small arteries, as they are the blood vessels traversing the subarachnoid space. That means if blood leaks out of these vessels (if these vessels bleed), the blood will be within the subarachnoid space between the arachnoid mater and pia mater. On CT, there will be **blood between gyri within sulci**—where CSF should be—that is restricted to a concave shape by the arachnoid-dura mater-skull; the blood fills the subarachnoid space, but not the space between the layers superficial (above) to the arachnoid. Between the blood now within the subarachnoid space and the brain parenchyma is merely a simple squamous epithelium, the glia limitans, and one

astrocyte's worth of pedicles. That is a wafer-thin barrier. And under the hydrostatic pressure of the artery, the subarachnoid space, usually supplied by the slow, gradual effusion of CSF from the choroid plexuses, overfills quickly. This pressure enables blood to affect the parenchyma. It doesn't go deep into parenchyma like an intraparenchymal hemorrhage (below), so it is classically described as **within the subarachnoid space** on CT. But blood is an irritant to the parenchyma, so affected individuals are stricken by an excruciating **headache** that has a rapid onset and crescendos quickly ("thunderclap"). Affected individuals complain of the "worst headache of their life." Saccular or "berry" aneurysms are commonly found in the anterior circulation, and represent a small defect in small arteries—there are no vascular smooth muscle cells in the tunica media, and the inner elastic lamina is lost. There is nothing to resist arterial pressures, and over time they worsen, until they leak or break. If they leak and heal, there is a **sentinel event** (a bad headache that went away. If they break, blood floods the CSF, and thus the patient presents with the **thunderclap headache** happening now. After the acute event is over, patients remain at risk for **vasospasm** (ischemic injury) and **seizure** in the days following the event. The presence of blood in the subarachnoid space can result in **fibrosis and scarring** of the arachnoid granules, predisposing the patient to **hydrocephalus** later in life. Aneurysms are associated with autosomal dominant polycystic kidney disease, coarctation of the aorta, and Marfan syndrome, among others.

Intraparenchymal hemorrhage represents two pathologies with two presentations, both of them hemorrhage into the brain parenchyma. When this is acute, independent of etiology, there is extravasation of blood; the parenchyma is pushed outward from the source of hemorrhage and compressed. The compressed tissue behaves as if there were a stroke, with complete loss of function of the affected area. This is also why the presentation of acute infarction gets a CT scan—to make sure it isn't an intraparenchymal hemorrhage. The surrounding parenchyma is edematous (cerebral edema). Eventually, the patient either dies or the edema and the hemorrhage stop, leaving behind hemosiderin-laden macrophages and regenerating astrocytes. Indeed, the same cellular events of ischemic insult are seen as the brain tries to heal. Old lesions, again regardless of etiology, show areas of **cavitation**—whatever the bleed pushed out of the way died, and when the clot was removed or degraded, nothing was left behind. One cause of intraparenchymal hemorrhage is **longstanding hypertension**, which leads to arteriolosclerosis of small vessels, leading to a weakening of the wall. These vessels are the lenticulostriate vessels (just see the words; we'll swing to this discussion in the Stroke lesson), resulting in lacunar infarcts. The cause of **lobar hemorrhages** (which can get so large as to cause an entire hemisphere to be a hematoma—hemispheric hemorrhage) is usually **cerebral amyloid angiopathy**, which leads to deposition of (we're keeping this vague on purpose) amyloid peptides in the vessel walls.

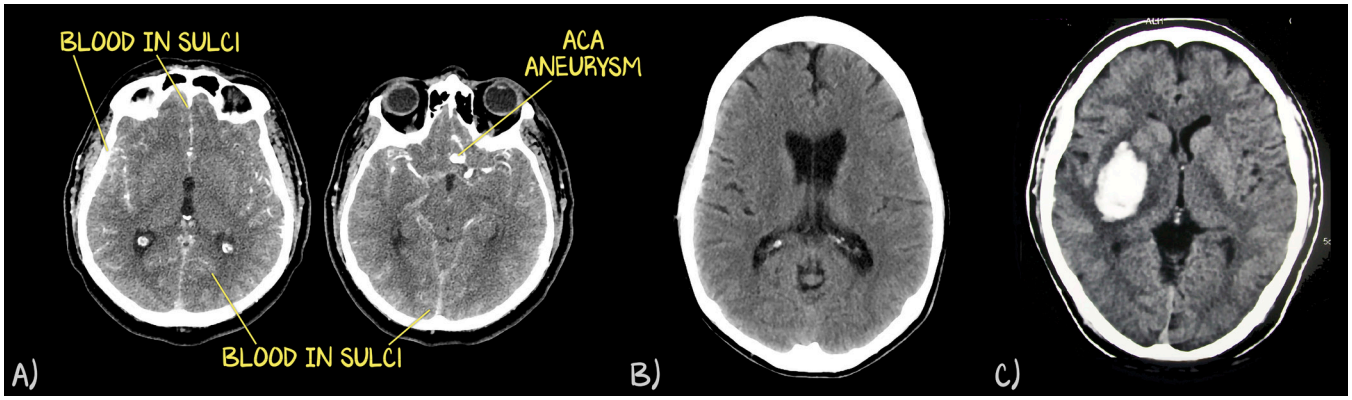

Figure 4.8: Visualizing Brain Bleeds, Part 2
(a) CT of a subarachnoid hematoma with blood (white stuff) filling the sulci. (b) A normal center CT for comparison. (c) CT of an intraparenchymal hemorrhage within the brain parenchyma, forming a rounded lesion. The lateral ventricle on the left side of the image is being deformed by the hemorrhage, as is the third ventricle.

The Cause of Increased ICP #3: Hydrocephalus

We covered obstructive and communicating hydrocephalus in the lesson on *The Flow of CSF: Ventricles and Sinuses*. We now bring them back briefly to talk about relieving ICP another way. Although the mechanisms above are either within the brain parenchyma or are bleeds that should never be there, hydrocephalus has a different mechanism with a different treatment. The CSF is supposed to be there; there is just now too much of it. Either acutely or chronically, a **ventriculoperitoneal** (VP, brain to the peritoneal cavity) shunt can be placed. The peritoneal cavity handles vastly more fluid per day than the ventricles can make. This VP shunt can be permanent.

You can't stick a tube into the brain parenchyma to drain the fluid because it is in between all the cells. You can stick a tube into a brain bleed, but you won't need to leave it there. This treatment just identifies another mechanism for addressing the cause of the ICP from a pathology you saw elsewhere.

Citations

Figures 4.4a, 4.4b, 4.4c, 4.5a, 4.5b, 4.5c, 4.7a, 4.7b, 4.7c, 4.8a, 4.8b, 4.8c: Courtesy of Radiopaedia.